been a stumbling-block in the way of all explanations of the histogenesis of locomotor ataxia. The frequency with which oculomotor paralyses occur in syphilis not attended by tabes seems to confirm the luetic theory.

In 1894 Edinger, of Frankfort, proposed a theory on the etiology of nervous diseases, and especially of tabes, which should not be disregarded. This theory is based upon the fundamental proposition that the molecules of a cell are in a state of labile equilibrium such that if one part becomes enfeebled, it is overthrown and strangled by its neighbors. In a similar manner components of tissues are hypertrophied at the expense of enfeebled constituents. When a cell performs its functions normally, its contents are destroyed, but are immediately replaced in the regeneration which occurs. If the cell be overexerted or enfeebled by toxins or other poisons, the connective tissue luxuriates, and we have an interstitial hypertrophy resulting. There is no question that in the normal performance of function, substances are destroyed which must be replaced. The difference in histological appearance and in reaction to stains between cells that have performed their function and those which have rested has been clearly demonstrated by Daschkiewicz, Hodges and Nissl.

Edinger presupposes an increased vulnerability of the spinal cord through the influence of syphilis, possibly through a toxin. He calls attention to the fact that of all nerves of the body, those which rule over the maintenance of the equilibrium are the most frequently called into action, and are those which suffer the greatest wear and tear in the ordinary functions of life. It is just these organs of coordination which are injured the soonest and the most severely in locomotor ataxia. Edinger ascribes this fact to his theory of diminished recuperation. According to him, the first thing that occurs in the luetic individual who is about to become tabetic is a diminished recuperation of the system of nervous elements that bring about coordination. In consequence thereof, the uninjured neuroglia tissue luxuriates, and a sclerosis results.

In the spinal cord, when an injury occurs at any one point, secondary degeneration of the whole system to which that part belongs results, and as a consequence we have the anatomical changes characteristic of tabes. This explanation renders it clear why tabes nearly always begins in the lower extremities and arises so frequently in those who exert the lower extremities to a great extent.

A careful examination of the mode of life of those who become affected with high tabes arising first in the upper extremities may lead to its explanation by this theory.

In a similar manner, Edinger explains the peculiar action of the pupils in locomotor ataxia. The pupils are normally constantly undergoing changes due to varying degrees of light, and the nervous elements involved in the reflex act are subject to constant strain. Through the diminished recuperative power, he presupposes the disturbance that we recognize as the Argyle-Robinson pupil results. Of the muscles around the eyeball, the levator palpebræ, the abducens and the rectus internus are the ones most frequently called into action, and as would be demanded by Edinger's theory, these are the ones most frequently involved in tabes.

It is by no means improbable that the atrophy of the optic nerve may be explained in a similar manner, and it will be of interest to determine to what extent eye-strain forms its etiological factor in the cases of locomotor ataxia in which it occurs.

Sanitation of Railway Cars in Kentucky was instituted in the latter part of 1904, and after some little trouble from the railway companies, the State Board of Health succeeded in enforcing its regulations. The supervision extends to cars of all sorts.

ACUTE DELIRIUM.*

By H. E. SANDERSON, M. D., Stockton.

THE closeness with which this affection is allied to the distinctly somatic affections met with in general practice, and the shortness of its course with lethal outcome, render it of more than passing interest to the profession at large, and entitle it to your earnest consideration at this time. When you remember that in the cases I am about to describe the patients were sent to us from considerable distances by rail, and in some cases from general hospitals, to die within a few days after admission, necessity of bearing in mind the existence and leading characteristics of the disease will be evident.

Status and Etiology. The present status of acute delirium is a much disputed point amongst alienists. It was recognized by the ancients who termed it phrenitis. In 1884, Luther Bell described it under the name of typhomania, since which time it has also been commonly called "Bell's Disease." Spitzka, in 1883, first used the term delirium grave. Other writers have failed to mention it as a clinical entity until recent times, and even now much confusion is caused by trying to place all cases manifesting delirium in the same category.

Coming down to the present time, we find a great diversity of views amongst writers. Clouston speaks of it as a phase of severe primary mania, and calls it delirious mania. He terms it "a further stage of acute mania," and takes issue with other authors in their prognosis of the affection. Kraepelin calls it a "symptom complex" and not an independent disease. He views it as a pathological condition developing in the course of various psychoses—especially dementia paralytica, mania, acute alcoholism, and active melancholia. He terms it an irritative brain lesion of sudden development soon passing into paralysis. But, while maintaining that similar symptoms may follow in the wake of pneumonia, myocarditis, and septicemia, he admits that it may occur as a primary brain lesion. The majority of French psychiaters, de Boismont, Ball, Brand and others, look on acute delirium as being an individual disease. Mendel, Furstner, Meynert, Schuele and Alzheimer regard it as a symptom complex. Dr. Soukanhoff is of the opinion that acute idiopathic delirium is by its evolution and course nearer in semblance to an infectious disease than to anything else. The same opinion was held by Briand, Bianchi, Puncinnino, and Rosari. Berkeley thinks it a matter of doubt as to whether it should be considered an entity or only a symptom complex. He says "in the few cases it has been my fortune to see, some diagnosis other than delirium acutum—principally post febrile or meningitic delirium—would have been equally justifiable," and cites the fact that similar symptoms occur after a host of infectious troubles—especially pneumonia, typhoid, typhus, phthisis, dysentery, measles, acute rheumatism, influenza, the puerperal state, and gastric cancer.

Kellogg regards it as a distinct form of mental disease, and says the term should be limited to those independent acute delirious manias which present the typhoid symptoms and run the hyperacute course described. He speaks of suppurative kidney and pulmonary disease, and surgical operations accompanied by prolonged anesthesia as causative factors while some cases can only be accounted for on the theory of perverted metabolism and auto-toxemia. Krafft-Ebing considers it a distinct affection due to "certain injurious circumstances acting upon the brain, primarily through the vasomotor nervous system, causing hyperemia, through paralysis of the vessel walls." He mentions long-continued hard struggle for existence as a common factor, though in a large majority of cases the patients are predisposed to nervous troubles and excessive vasomotor irritability. Schuele

* Read before the San Joaquin County Medical Society, December, 1904.

has known it to result from excruciating physical suffering. In most cases it was noted that the patient —usually between 20 and 40—who has for a long time been in a feeble state of health, suffering from malnutrition or nervous prostration, (due frequently to poverty or drunkenness) experiences some extra strain on the nervous system. Such a strain might occur from emotional shock, due perhaps to an unhappy love affair, alcoholic excess, business crisis, or the puerperal state, which would precipitate the outbreak of delirium. Abandonment of seduced and pregnant girls is a prominent element in the history of some cases. The recorded cases of "meningitis from overstudy" are in reality instances of acute delirium, and are brought about as much by the emotional strain attendant upon competitive examinations as by the mental effort itself. Other assigned causes are the menopause, insolation and trauma. With such causes, an instability of the central nervous system would be induced; and the presence of an irritating and debilitating poison, whether from within or without, would more easily overturn the mental equilibrium.

Spitzka says: "It is never a strong mind nor a healthy body that suffers in this way, but one inherited from a feeble ancestry, and our school system is not to be blamed for it." Macpherson, while admitting the fact that acute delirium occurs sometimes in predisposed persons as a consequence of profound physical and nervous prostration, and malnutrition, considers that it is the immediate result of bacterial toxins, and places it in the list of "forms of insanity resulting from autointoxication or microbic toxins." A number of investigators have described bacteria found in the urine, blood and other parts of the organism, in cases of acute delirium. Sir John Baty Tuke described a long, rather thick, bacillus with numerous spores found in the urine and blood. Babcochi found in the cerebrospinal fluid prior to death the micrococcus pneumoniæ crouposæ and streptococcus pyogenes aureus, the former in great numbers, the latter in scattered chains. Ceni found in a series of experiments that the blood of the insane suffering from intense motor unrest, and that of animals whose muscular systems had been severely exhausted by the continuous application of faradism, form a favorable culture medium for the growth of bacteria. One must, however, remember that in the blood of the insane generally, the presence of pyogenic organisms has been frequently determined.

Bianchi obtained from the blood of patients with acute delirium a certain organism, having the form of a bacillus, two or three times as long as broad, and with tendency to unite in chains. It was mobile, stained with the ordinary aniline colors and by the Gram method and was non-sporific. He cultivated it in agar and in broth. Various forms of cocci, specially the streptococcus and staphylococcus were also found in large numbers. He regards the bacillus as peculiar to the affection and has found it in autopsies in the subarachnoid and ventricular fluids. Ceni found only staphylococci, and believes the microorganisms found are only of secondary importance.

The various researches on the bacterial origin are seen therefore to be by no means in accord; nor indeed could accord be expected, inasmuch as the inciting sources of the disease are so manifold. Some of the cases described as acute delirium have been either aggravated phases of a primary psychopathy, or the expression of other intoxications. We are warranted in inferring that certain cases are due to inoculation with the toxin productive of primary and genuine acute delirium upon ground already occupied by other psychopathies. We should not include under acute dilirium, mere episodes of other mental troubles which somewhat simulate it.

The following cases occurred at the Women's Department of the Stockton State Hospital during my term of service, beginning January, 1895.

L. W. Age 45, widow, keeper of boarding house, American. Admitted March 16, 1895. Family history unknown. Before admission for five days was boisterous, very talkative and violent. Ran away from her home and refused to return. History otherwise unknown. March 18—Noisy, incessantly talking and incoherent. Refuses food and drink. Has profuse muco-purulent expectoration. Always expectorates on the floor. March 21—Has not changed since the 18th. No rise of temperature; respiration normal; pulse 100, normal in frequency and strength. Physical examination negative. Bowels constipated. Mouth has sordes on tongue, lips and teeth. Is delirious. April 13—Patient has been delirious since last report. Died today. No postmortem allowed.

A. W. Age 20, single, dressmaker, American. Admitted April 29, 1897. Family history unknown. Before admission had previous attacks of insanity; nature not given. Has been insane for two years. Imagined she had a man and a baby in bed with her, and that she was in some strange place, while at home. Has repeatedly threatened suicide. Is said to have been subject to hystero-epileptic convulsions. Irrational and incoherent; hallucinations of sight. On admission, is prostrated; tongue dry and coated; sordes on teeth; delirious; slight eruption of acne in places. Physical examination negative. Is noisy at times and picks at the bed clothes. May 1—Is in a stuporous condition. Occasionally screams. No fever. Respiration 30; pulse 90. Physical examination negative. May 3—Lies in a stupor all the time. Screams at times. No apparent hallucinations. Pupils equal and react well. Tongue dry and coated. Cannot be roused. Respiration 46; pulse 100. No rise of temperature over 100 since here. May 4—Looks worse. Temperature 102 F. this morning. May 7—About the same as when last recorded. Temperature has not risen over 101½ since yesterday. Respiration 40; pulse 100. Physical examination negative. In a stupor all the time. Receives considerable stimulants and nourishment. May 9—Patient died. No postmortem permitted.

B. C. Age 28, married, housewife, Austrian. Admitted May 3, 1900. Family history: Father had epilepsy. Before admission, attack began April 30, 1900. Claimed her husband put the children into the fire; also that he sold their little girl of nine years. Imagines the husband wants to kill her and that he is in love with another woman. She jumped out of bed and ran into the street cursing the neighbors. May 4—Was noisy when she first came. Is now irrational, incoherent, excited, violent and frightened. Talks to herself; kicks and bites. Pupils dilated and active. May 5—Looks worse. Has an herpetic eruption on the lips; sordes on the teeth and tongue. Breath foul; pale; pinched face. Temperature reached 103° F. Physical examination negative. May 7—Died.

Postmortem examination. Meninges congested; lymphatic engorgement in the pial vessels with opaque appearance. Pia somewhat adherent to cortex. Brain tissue much congested. Brain swollen, softer than normal, and edematous. Large amount of fluid between pia and brain and in the ventricles. Punctate extravasations in brain substance; light pinkish appearance in cortex. Injection of vessels extends into the spinal cord. Dura mater adherent to skull. Lungs show hypostatic congestion. Heart apparently not affected. Abdominal cavity quite dry; intestines empty and appear much contracted. Other organs somewhat congested.

L. M. Age 38, single, dressmaker, American. Weight, 200 pounds. Admitted May 29, 1901. Family history: One sister was insane. Before admission, destroys her clothing; tried to run out into the hallways of the building where she roomed. Will not talk. Imagined that men were coming and going from her room at all hours and that the house in which she lived was not respectable. Defecated on the floor. Attack began one week ago.

May 30—Is restless; stubborn; moans occasionally. Temperature 101° F. this morning. Pulse 90, full and regular. Quiet; no delirium. Will not talk except occasionally to reply briefly to questions. Lies down and rolls on the floor; refuses food; has to be restrained. June 1—Has temperature 102° F.; pulse 100; respiration 24. Physical examination negative except systolic murmur over mitral and aortic valves. June 2—Condition about the same. Temperature reached 103° F.; pulse 100; respiration 28. Tongue dry and coated; sordes on tongue, lips and teeth. No delirium. Vacant expression. Speaks only a few broken sentences. No hallucinations nor delusions apparent. In a stupor. June 3—Delirious. Temperature 102½; pulse 140; respiration 30. June 4—Temperature 102; pulse 130; respiration 36; stuporous. June 5—Temperature reached 104. Died. No postmortem permitted.

G. P. Age 45, widow, domestic, French. Admitted July 28,1902. Family history unknown. Before admission, was noisy, turbulent, irrational in speech and actions. Hears voices and imagines that people are after her. Disrobes;, threatens suicide; wishes to kiss everyone; pulls her hair.

July 29—Temperature 101° F. Pulse 120 intermittent. July 31—Will not eat. Thinks her food is poisoned. Is violent; frequently attempts to disrobe. Temperature 100° F.; pulse 140. Restless, noisy; talks disconnectedly. August 1—Restless; noisy; sleepless; delirious. Pulse 144; temperature 103½° F. August 2—Condition the same as yesterday; temperature 104° F.; pulse 144. August 3—Temperature 103° F.; pulse 120. August 4. Mind clearer. Talks a good deal. Temperature 101½° F.; pulse 129. August 5—Died 2 A. M.

Postmortem examination. Dura mater adherent to skull over the frontal lobes. Pia mater congested; lymphatics turbid in places. Brain wells out on removing skull; large increase of fluid; brain softer than normal and much congested, showing hemorrhagic points on section and pinkish appearance. Viscera normal except congestion of kidneys. Liver pale. Marked dryness of abdominal cavity. Stomach congested especially at greater curvature.

J. N. Age 26, married, domestic, French. Admitted February 7, 1903. Family history unknown. Before admission, was in one of the leading hospitals of San Francisco for three weeks and sent from there to us. Patient entered there in a delirious state. Temperature 98.4° and pulse 92. On third day temperature was 99° and pulse 100. From then till end of first week temperature was one degree below normal. The second week she appeared well and acted rationally. At beginning of third week, again became delirious and maniacal, with hallucinations of sight and hearing. Beat her head against the bed, when not restrained, and attempted to jump out of a window. Temperature and pulse rate could not be obtained that week because of resistance. At the beginning of the fourth week (February 7, 1903) she was sent to this hospital. On admission here, temperature reached 100; pulse 80, and very weak; respiration 18. Face flushed; pupils widely dilated; lips dry and cracked; teeth covered with sordes; tongue dry and brown. Very restless. Tried to bite the attendants whenever they came near her. Seemed in great fear; resisted everything. Talked incessantly and incoherently, often repeating certain words. Refused food.

February 8—More restless. Makes automatic motions with the hands; has facial twitchings. Becoming more prostrated, and rapidly emaciating. Sleepless. Low muttering delirium. Such was in brief the course of the patient's symptoms during her five days with us. The temperature on February 8th reached 102½° F.; February 9th, 102½° F.; February 10th, 104° F.; February 11th, 104½° F., and a few hours before death on the 12th, 106° F. Died February 12th.

The treatment employed was mainly rest, quiet, nourishment, spongings, laxatives, and hypodermics of ergotin. Urine (obtained by catheter) showed small amount of albumen, a few casts, heavy deposits of urates; specific gravity 1025. Physical examination of chest and abdomen were negative.

Postmortem examination. Brain much congested and edematous. Largely increased amount of fluid between meninges and brain, especially collected over frontal lobes, flattening them somewhat. Pia somewhat adherent in places; course of lymphatics marked by white streaks. Sections show scattered hemorrhagic puncta; cortex has light pinkish appearance; congestion of brain extends down into the spinal cord. Rest of the body not examined through lack of permission.

Clinical Delineation. The typical course of the affection is as follows: A preceding history of prolonged mental or physical over-strain, or of alcoholic or sexual excesses. Prodromes of restless anxiety, despondency, forebodings of evil, general malaise, and disturbed sleep. Next, abrupt maniacal explosion, with great violence of motion, incoherence, boisterousness, gesticulations, vivid hallucinations. Then remissions of a few hours may occur. In a day or two, great exhaustion, automatic activity, delirious ideation, obscuring of consciousness, muttering, subsultus tendinum. Temperature rises to 102°F. or even 106°; pulse frequent and feeble; sordes on lips and teeth; tongue dry, brown and heavily coated. Semi-coma; convulsive movements, involuntary rectal and bladder evacuations; rapid emaciation and finally death within fourteen days of the first appearance of maniacal symptoms. Rarely the end may come in thirty-six hours or it may be delayed till the end of the third week.

In cases not resulting fatally, the comatose stage is not reached; the violence abates, and the patient is left exhausted in mind and body. After many weeks recovery may occur, but in one-half of these non-fatal cases, a partial recovery only is obtained, and the patient remains more or less demented.

Differential diagnosis. Concerning the differential diagnosis, it is necessary to distinguish acute delirium from:

1, acute alcoholism; 2, acute meningitis; 3, acute mania; 4, delirium of fevers; 5, paretic dementia.

1. In acute alcoholism, single symptoms may be similar, but the temperature is higher in acute delirium and the reduction of consciousness is more complete. The alcoholic tremor is wanting, though a coarse tremor exists in exceptional cases. When alcoholic excess has been an exciting cause the difficulty is greater. The motor symptoms of acute delirium are more those of cerebral irritation than of a mere psychical disturbance such as occurs in alcoholism.

2. In meningitis there is a very acute beginning, frequently an initial chill; early appearance of sopor, convulsions, stiffness of neck muscles, opisthotonos, general hyperesthesia, and less marked remissions—also may have well-marked paralysis. There are confusion of ideas and delirious excitement in both diseases, but the motor agitation is not so great and the reduction of vital force is not so sudden in meningitis.

3. Maniacal and melancholic frenzy are preceded by the ordinary and readily recognizable symptoms of those psychoses; while acute delirium is either sudden or preceded by a state of impaired consciousness of a kind not found in mania nor melancholia. The ideation is much more incoherent and shows either a frightened or an angry state. Speech rapidly deteriorates and the patient is finally unable to pronounce syllables. In mania there is no considerable rise of temperature nor acceleration of the pulse; and there is loquacity with a ready flow of ideas in place of inhibition or monosyllabic repetition. There is also not the increasing stupor nor hallucinatory-confusional delirium and not the general wasting found in acute delirium. Death in acute mania is rare, while very frequent in acute delirium. Even in the confusional form of acute mania there is not the rise of temperature; while obtundity alternates with prolonged periods of loquacious excitement.

4. Delirium of fevers. In pneumonia the delirium lacks the motor violence and is relieved by antipyretics. The dilirium of fevers in general is not of such an intense character and there is a difference in the temperature curve, in some cases a typical rash, together with a sequence in other symptoms unlike that of acute delirium. This is notably the case with typhoid fever.

5. Paretic dementia. In the galloping or so-called fulminating form of paretic dementia, there is a previous history of character changes or changes in mental disposition for months previous. Delusions of wealth and power; erotic ideas; hallucinations of fire and bloody scenes together with a general persecutory delirium, are here present. The temperature seldom rises so high and is more constant. Remissions do not occur with the same regularity in paretic dementia.

Pathology. The pathological processes resemble those of toxic or septic conditions. At first intense congestion of cerebral regions and following the hyperemia, venous stasis with edema. Effusions of blood corpuscles and leukocytes occur in the perivascular spaces, and the ganglionic elements are swollen or in process of disintegration. Sometimes punctate extravasations of blood in the brain substance; lymphatic engorgement; membranes injected and adherent; lungs hypostatic congestion or edema; sometimes lobular pneumonia; heart lax and contains dark fluid blood; muscles soft and pale from fatty or granular degeneration; blood dark and watery; liver, spleen and kidneys engorged; muscles atrophied; micro-organism at times in blood and urine; the hyperemia of the brain may extend to the cord; brain so edematous that it wells out of the skull cavity; cortex appears swollen and dotted with punctiform hemorrhages; the large vessels of the pia appear as white stripes; general musculature granular with wax-like degeneration. On the whole the condition of the brain points to a very low tone in the vessel walls, followed by a hyperemia of the vessels, while the vessels are apt to be varicose and tortuous. The basal ganglia are involved. Local areas of encephalitis exist. Transuded white corpuscles fill the lymph sheaths. Veins and arteries are packed with blood corpuscles. Plastic exudate forms in the spaces. Shrinkage of cells of brain occur and they are pressed upon by pericellular

coagulated exudate. Similar appearances, though less marked, are found after chemical intoxication—psychoses, collapse delirium of fevers, and more rarely after alcoholic delirium. In fact, the whole appearance might be described as that of infectious hemorrhagic encephalitis.

Prognosis. Prognosis is bad. Some few patients recover, but of these a large percentage are incomplete restorations, and show more or less evidence of permanent dementia. Most patients die within two weeks.

Treatment. The treatment is symptomatic. Nothing is of much benefit. Some claim good effects from ergotin injections. The patient should be kept in a large, well-ventilated, darkened, and rather cool room. The temperature of the room should not be over 60°, the patient kept in bed by a restraining sheet, and constipation relieved with croton oil, which also tends toward derivative effect. Reduce temperature by cold sponging or packs. Give as much nourishment as possible, including cool drinks, especially milk. Chloral hydrate should be avoided because of its tendency to induce cerebral hyperemia; and hyoscine because of its depressing effect. Morphin may occasionally be needed for allaying excitement. Bromides are useless. Hypnotics are also of no avail. Bleeding and counter irritation are not recommended. Alcoholic stimulants are needed when heart weakness appears. Care must be taken to avoid decubitus.

The number of admissions into the Women's Department of our hospital during the years covered by this investigation, namely from January 1, 1895, to March 1, 1903, was 864. This would make an average of one in 108 admissions. During each of these years, we have had one case of acute dementia, except in 1899, when there were none. In looking over the statistics of a number of the hospitals for the insane elsewhere, I find such a variation in the reported proportion of cases of acute delirium, that I am forced to the conclusion that the statistics have depended very largely upon the diagnostic acumen of the compilers.

Of fifteen typical cases described by A. S. Rowley of the Northern Michigan Asylum, in the *American Journal of Insanity*, six had insane relatives, and only three were known to have none such. Only two had no previous history of insanity. One had had epilepsy for twenty-three years. Only one appeared well nourished. Of the fifteen, only three survived. In reviewing our cases we find one almost the counterpart of each of the others, both as to symptoms, duration, termination and postmortem findings, when such could be made. Cases have been reported by others, in which no physical indications of a definite somatic disease during life, the necropsy showed deep-seated lobar pneumonia, in addition to the hyperemia, lymphatic engorgement and edema of the brain and meninges; so that such a possibility is not excluded in cases not followed by a careful necropsy, though it is decidedly exceptional.

SUPPORT THE BOARD OF EXAMINERS.

"These facts show why it is the doctor's duty to the state to support the medical examining boards, to work to have honest men appointed upon them, and not to be too censorious of their shortcomings. We, who have not served on these boards, know little of the worry, the work, and the weariness entailed by honest service in them. The man, who unreasonably or unjustly decries the system and its exponents, is doing an economic wrong similar to that of those few honorable but short-sighted doctors, who for years played into the hands of the profession's enemies by opposing state control of medical licensure. The manner, in which state laws compelled low-grade medical colleges to adopt entrance examinations, lengthen terms, and exact efficient final examinations, has fully justified the prophecies of the advocates of state control."—John B. Roberts, Philadelphia.

COMMUNICATIONS.

AN APPEAL TO THE GENERAL PRACTITIONER.

To the Editor of the STATE JOURNAL: Perhaps when you were a school-boy and less venerable in appearance than at this present date, you occasionally heard some of your companions spouting on "declamation day," the familiar lines: "Why is the forum cr-r-owded? What means this stir-r-r-in Rome?" If so, you may be interested in another "stir" now agitating the camp of the California opticians.

Some two years ago certain of the grinding folk got a bill through the Legislature establishing a board of examiners in "Optometry" (sic). When it came into the hands of our present governor, he, it is said, signed it reluctantly while remarking (he is, as you know, an oculist): "Gentlemen, the day will come when you will regret the passage of this bill."

I am in possession of reliable information and personal knowledge which proves that the governor has been a true prophet, and that the better class of opticians now bitterly regret the success (?) of those of their craft who engineered the bill.

The real animus of this bill is apparent: To give at least a *quasi* professional status to its originators in the eyes of a public as yet uneducated regarding what a *prescribing* optician does not know. The Board of "optometrists" (you will not find the name in any dictionary), began to grind; this time upon raw opaque material of unusually inferior quality, and so industriously, that the army of opticians is now crowded with raw, half-baked recruits legalized to prey upon the eyesight of the credulous and ignorant. *Hinc illæ lachrymæ*, issuing from the eyes of the "legalized." Our brethren of the Empire State have thwarted successfully, several efforts of the opticians there to achieve identically pernicious legislation through "The 'Optical' Society of the State of New York." In a recent open letter, Dr. Frank Van Fleet, Chairman of the Committee on Legislation of the Med. Society of New York, states that the Society opposed, during the legislative session of 1904, a petition originated by the above "Optical Society" for a law creating a state board of examiners in "optometry." He writes:

"At the time of the hearing on the optometry bill before the legislative committee of last year, the opticians presented a long list of names of physicians who had endorsed their efforts. * * * The undersigned communicated with every one named on the list, and learned that where reputable physicians had endorsed the measure it was through a misapprehension of the real purpose of the bill; and when its true character was pointed out to them, they not only withdrew their indorsements, but in many cases wrote vigorous letters in opposition to it. Many of the names were fictitious, the communications addressed to the addresses given being returned as not found. A large number were the names of irregular practitioners, such as osteopaths, spiritualists, etc. The arguments presented by the opticians are very misleading. Their claim, of course, is that they desire to protect the community from incompetent people, but the fact is (*as every well-informed physician must know*), [italics mine] they are all incompetent." —*New York and Phila. Med. Jour.*

I invite your especial attention to this closing sentence, for it is my personal experience that the prescribing optician gets his most powerful "boost" from the "well-informed" but alas! inconsistent physician who goes to the optician for his own glasses! This same "well-informed" doctor would scorn an oculist who went, when ill, straight to a druggist for *advice with medicine thrown in on the side.*

To do justice to the better class of opticians we may heed the saying: "Live and let live;" 'tis an old saw and a just one, so we should not lump the "optometrist" and like "ists" with the reputable and conscientious optician, who, when he finds he cannot bring his customer's vision to normal, generally tries to persuade him to consult an oculist.

But while there is a difference in these two specimens of amateur doctors, woe to those who have incipient tabes or albuminuria, etc., and who go first to either of these amateurs not knowing he has a serious disease of which the deficient vision is but a symptom.